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MICROSCOPIC EXAMINATION OF ORAL SINUS TRACTS AND THEIR ASSOCIATED PERIAPICAL LESIONS

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ABSTRACT

Microscopic examination of 30 sinus tracts revealed that 100% were lined with epithelium at the surface mucosal interface. Twenty of thirty (67%) sinus tracts did not have epithelium deeper than the surface mucosal rete ridges. The remaining extension of the sinus tract was lined with granulomatous tissue. Ten of thirty (33%) sinus tracts had epithelium extending down the tract to the periapical lesion. The periapical inflammatory lesions that the sinus tracts communicated with were microscopically diagnosed as abscesses, granulomas and cysts.

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Harrison and Larson (1) have pointed out the confusion there is concerning the nomenclature and histology of sinus tracts. As stated in An Annotated Glossary of Terms in Endodontics (2), the term sinus tract "refers to a tract leading from an enclosed area of inflammation to an epithelial surface." It also states that the term dental fistula "should be discouraged, and the more proper term sinus tract should be used." In 1961, Bender and Seltzer (3) reported that they found sinus tracts to be lined with granulation tissue not epithelium. In 1965, Ingle (4) described a photomicrograph of a sinus tract as having a sheath of chronically inflamed connective tissue. Two other photomicrographs were used to illustrate sinus tracts fully lined with epithelium. The absence of an epithelial lining was supported by Grossman (5). After examining a number of cases histologically, he found no evidence of an epithelial lining in sinus tracts. Smulson (6) believed that sinus tracts were usually lined with granulomatous tissue, but could be lined with stratified squamous epithelium or ciliated columnar respiratory epithelium depending on the surface surrounding the stoma. Ingle (4) published a photomicrograph with ciliated epithelium in a sinus tract draining to the maxillary sinus.

In 1973, Valderhaug (7) microscopically examined 13 experimentally produced sinus tracts in monkeys. When the pulp was extirpated and the teeth left open for less than 100 days, sinus tracts did not develop. When the teeth were left open for 100 to 200 days, 6 sinus tracts developed. None of these tracts was lined with epithelium. When a group of 16 teeth were left open more than 200 days, 7 sinus tracts developed. Four of the seven tracts were lined with epithelium.

In 1976, Harrison and Larson (1) reported a histologic evaluation of ten sinus tracts surgically removed from humans. Only one sinus tract was lined with stratified squamous epithelium. The remaining nine sinus tracts were lined with granulation tissue that contained chronic inflammatory cells. The known duration of the epithelium-lined tract was two years. Because other long-standing sinus tracts were lined with granulation tissue, they concluded that there was no correlation between duration and histologic appearance of the lining of sinus tracts.

In 1982, Vire and others (8) reported a case that included a photomicrograph of an epithelium-lined sinus tract that had a known duration of nine years. They felt that chronicity may be related to epithelialization.

Several (3,4,7,8) of the earlier microscopic evaluations of sinus tracts used longitudinal sections to examine the specimens. Because of the tortuous nature of sinus tracts, the presence or degree of epithelialization can be difficult to interpret with longitudinal sections. Vire and others (8) encountered this problem in their recent case report. Initial tissue sections showed an epithelium-lined sinus tract that seemed to come to a "dead end". Deeper sections demonstrated that it was a tortuous epithelium-lined sinus tract throughout its length. Harrison and Larson (1) recommended serial sections at right angles to the long axis of the sinus tract. This technique should avoid sampling error and show the true extent and nature of the variable lining of a sinus tract.

The purpose of this study was to microscopically examine closely spaced sequential cross sections of sinus tracts removed in toto

from the mucosal surface to the periapex and to determine the nature and structure of their lining. In addition, the periapical lesions to which the sinus tracts communicated were microscopically examined in half the cases.

MATERIALS AND METHODS

Biopsy specimens of 15 sinus tracts and their associated periapical lesions were obtained from extracted non-restorable teeth. An additional 15 sinus tracts without the periapical lesion were also obtained and microscopically examined. The pathway of each sinus tract from its stoma in the mucosa to the periapical lesions was confirmed radiographically by the passage of a gutta-percha cone through the sinus tract. With the gutta-percha cone left in place, the sinus tract and a margin of surrounding tissue was dissected out along the sinus pathway from the mucosal surface to the periapex. All specimens were immediately fixed in 10% formalin. The sinustract specimens were oriented for sequential sectioning using the gutta-percha cone as a guide (1). The specimens were processed with the gutta-percha cone left in position. Cross-sectional cuts were made at 50 mu. intervals beginning at the mucosal surface and extending to the periapical end of each specimen. Using this technique, it was possible to gain a three-dimensional appreciation of the structure of a sinus tract and to follow the sinus-tract lining throughout its course (Fig 1-4).

RESULTS

All 30 (100%) of the microscopically examined sinus tracts were lined with stratified squamous epithelium at least to the level of the mucosal rete ridges.

In the first group of 15 sinus tracts, 5 (33%) were lined with stratified squamous epithelium the entire length of the tract. The other 10 (67%) of the sinus tracts were lined from the level of the rete ridges to the end of the tract with granulomatous inflammatory tissue infiltrated with both acute and inflammatory cells (Table 1).

An identical distribution was found in the second group of 15 sinus tracts biopsied with their associated periapical lesions. Once again, 5 (33%) sinus tracts were lined with epithelium the length of the tract, and 10 (67%) were lined with granulomatous tissue from the level of the rete ridges to the end of the tracts. The patient's tooth number, position of sinus tract, size of apical rarefaction, periapical microscopic diagnosis and presence or absence of epithelial proliferation is given in Table 2. (The duration of the sinus tracts is not reported because most patients were either not aware of the sinus tract's presence or were unsure of its duration.)

When both groups are combined, 10 out of 30 microscopically examined sinus tracts had epithelium extending along the tracts. The remaining 20 sinus tracts were lined with granulomatous tissue.

In all 30 sinus tract specimens, the orifice of the sinus tract was lined with epithelium from the surface mucosa. In 20 (67%) of these specimens, the epithelium stopped near the level of adjacent rete ridges (Fig 5). From this point to the apical inflammatory lesion, the sinus tract was lined with granulomatous tissue containing both acute and chronic inflammatory cells. In 10 (33%) of the sinus tract specimens, the epithelium extended down the sinus tract as either a complete or

interrupted lining to the periapical lesion (Fig 6).

Of the 15 periapical lesions examined, 4 were determined to be periapical cysts. In two cases, the cystic epithelium appeared to merge with the sinus tract epithelium (Fig 7). In no instance was the cystic epithelium seen to migrate into an otherwise non-epithelial lined tract (Fig 8).

Of the 15 periapical lesions that were microscopically examined, there were 4 periapical granulomas (chronic apical periodontitis), 4 periapical cysts, and 7 periapical abscesses (Table 2).

Only 2 of the 15 biopsied periapical lesions had a radiolucency smaller than 5 mm. in diameter. They were both over 3 mm. in diameter (Table 2).

DISCUSSION

A sinus tract usually develops as a route of drainage from a periapical inflammatory lesion and follows a path of least resistance through bone, periosteum and mucosa. Formation of a patent sinus tract may be preceded by a subperiosteal abscess with eventual drainage of the inflammatory exudate through the stoma of a sinus tract. The stomas are usually in close proximity to their source of drainage although at times the tract may be some distance from the source. In 1917 Partsch (9), as referenced by Mortensen and others (10), found in a group of 758 sinus tracts that 400 were from the maxilla and 358 were from the mandible. In their own study, Mortensen and others (10) examined 1600 teeth with periapical lesions and found 136 (8.5%) had sinus tracts. Of these sinus tracts, 119 opened on the facial surface, 8 on the palate, 2 perforated the mandibular lingual sulcus, and 7 were

found extraorally. In our group of 15 sinus tracts (Table 2), all but one were found on the facial surface. One sinus tract from the lingual root of an upper molar was found on the palatal surface.

All 30 sinus tract specimens had the surface orifice lined with epithelium from the surface mucosa. A few microscopic sections showed rete ridges confluent with the sinus tract at a superficial level. They may be the source of some epithelial cell proliferation near the surface.

In addition to surface mucosa, it seems possible that the source of epithelium in a sinus tract may be from rests of Malessez or from periradicular cysts. Four of the 15 periapical lesions in our sample were determined microscopically to be periapical cysts. Two of these periapical cysts communicated with sinus tracts lined with epithelium and two with sinus tracts lined with granulomatous tissue. Although it is possible that the radicular cysts were the source of some epithelium in the two sinus-tract specimens that were lined with epithelium, it was our impression, because of the greater thickness of the epithelial lining and its tapering growth in a periapical direction, that the epithelial lining originated from the surface mucosa. None of the specimens demonstrated only epithelial extension from the periapical lesion into the periapical end of the sinus tract. Valderhaug and others (7) did find isolated strands and islands of epithelium in the apical area of sinus tracts experimentally produced in monkeys. They assumed that, unless the epithelium was destroyed by inflammation, it would eventually merge with epithelium from the oral surface.

In the past, some authorities have indicated that the presence of epithelium in a sinus tract necessitates special treatment. Sommer and others (11) recommended that sinus tracts be cauterized with phenol to remove the epithelium. Bhasker (12) believed that if a "fistula" is lined with epithelium, root canal therapy must be accompanied by an apicoectomy. Curettage of the sinus tract was not specified. Grossman (5), however, has maintained that a "fistula" required no special treatment and would close as soon as the root canal is adequately cleansed, and that only in cases of nonclosure should the tract be curetted. Today, most authorities agree with the latter. Stromberg and others (13) reported complete healing of 26 sinus tracts when the only treatment was non-surgical endodontic therapy of the involved teeth.

Because the orifices of all sinus tracts are lined with epithelium to about the level of the rete ridges, they have a similar clinical appearance and it is impossible to clinically determine if a sinus tract is lined with epithelium past its stoma without surgical removal and microscopic examination of the sinus tract. It would thus seem that as Harrison and Larson (1) state, the significance of epithelium lining in a sinus tract will remain obscure until a clinical means of detection can be devised.

As long as a sinus tract remains patent, clinical symptoms are minimal or non-existent. The tooth in question may not be sensitive to percussion and palpation, but the patient usually will relate a prior incident of discomfort. If the sinus tract becomes blocked, then symptoms may arise. It is believed that the inflammatory discharge

increases if there is an increase in quantity of irritant from the canals, an increase in virulence of microorganisms, or a decrease in patient resistance (4). Histologically, a delicate interplay between acute and chronic inflammation seems to take place.

Several sources (4,14-16) state that the lesion develops from a chronic apical periodontitis (granuloma) (2) or an acute apical abscess (2). Ingle (4) believes that the development of a "suppurative apical periodontitis" from an acute alveolar abscess is seldom the case. That seems to be supported in this study and previously by Mortensen and others (10) because the size of the periapical radio-lucencies are consistent with a chronic apical periodontitis. With an acute apical abscess, all that is usually radiographically visible is a slight widening of the periodontal ligament space (4). Mortensen and others (10) found that only 5% of the teeth with periapical radiolucencies less than 5 mm. in diameter were associated with sinus tract. However, 19% of the teeth with periapical radiolucencies over 5 mm. in diameter exhibited sinus tracts. In our group of 15 sinus tracts, 13 of the periapical lesions were 5 mm. or more in diameter (Table 2).

Seven of the 15 microscopically examined periapical lesions were consistent with periapical abscesses. Four of the 15 periapical lesions were determined to be periapical cysts and 4 other periapical lesions were determined to be periapical granulomas. Although the latter 8 lesions were suppurative in nature as evidenced by the patent sinus tracts, the tissue sections that were microscopically examined did not contain a circumscribed collection of pus in the tissue which is indicative of abscess formation (2). As mentioned by Smulson and others (16),

the zones of a periapical abscess may be similar to a periapical granuloma except for the amount of pus around the apices. Sequential sections of the periapical lesions may have demonstrated areas of liquefaction necrosis. In addition, subjective histopathologic interpretation of inflammatory lesions may be difficult if the lesion is changing character. Lalonde (17) put almost 10% of his periapical specimens in a "miscellaneous granulomatous lesions" category that includes early cystic transformation, abscess formation, foreign body reaction, and scar formation. Seltzer and others (18) have demonstrated that the proliferation of epithelium is associated with the presence of periapical inflammation. Cyst cavity formation may occur in an apical granuloma when there is degeneration of the cells in the center of an epithelial mass or by epithelium proliferating and covering the connective tissue surface of an abscess cavity (19).

The terminology describing the periapical lesion associated with a draining sinus tract has been very inconsistent. Suppurative periodonitis (chronic abscess) (20), acute apical abscess (11), chronic apical abscess (11), chronic alveolar abscess (5), suppurative apical abscess (4), granuloma with liquefaction necrosis (14), chronic suppurative apical periodontitis (15), suppurative apical periodontitis (4), and chronic periapical abscess (16) are terms that have been used in endodontic textbooks.

Because of the dynamic nature of periapical inflammatory lesions, a definitive diagnosis based solely on microscopic interpretation can be difficult. Morse and others (21) characterize a granuloma as a

localized collection of granulomatous tissue in the periapical tissues. In their opinion, several periapical inflammatory lesions are variations of a granuloma. They consider a chronic alveolar abscess as a granuloma with liquefaction necrosis. They described this lesion as "a diffuse accumulation of granulomatous tissue in which pus has formed and often a sinus tract (fistula) develops."

They also characterize a periapical cyst as a derivative of a granuloma in which epithelium has proliferated and lines a sac. Morse and others' (21) classification seems appropriate for the periapical inflammatory lesions associated with the sinus tracts that were examined in this study.

SUMMARY AND CONCLUSIONS

Thirty oral sinus tracts that communicated with periapical inflammatory lesions were microscopically examined. In 15 of those cases, the periapical lesion associated with the sinus tract was also microscopically examined. Within the parameters of this study, the following conclusions were made:

- (1) All 30 (100%) of the microscopically examined oral sinus tracts were lined with stratified squamous epithelium at the surface mucosal interface. Thus, clinical appearance cannot be used to determine if an epithelial lining extends to the periapical lesion.
- (2) Twenty of 30 (67%) sinus tracts did not microscopically demonstrate epithelium deeper than the surface mucosal rete ridges. In these specimens, the remaining sinus tract was lined with granulo-matous tissue.

- (3) Ten of 30 (33%) sinus tracts microscopically show epithelium extending the length of the sinus tract to the periapical lesion.

 The epithelial lining may be either complete or interrupted as it extends down the sinus tract.
- (4) In no case did the epithelial lining of a sinus tract communicate only with radicular cyst epithelium and not with surface mucosal epithelium. In this study, the epithelium lining of the examined sinus tracts seemed to originate from epithelium at the mucosal surface and not from proliferating epithelium of the periapical lesion.
- (5) A sinus tract may be lined with either granulomatous tissue or epithelium, and it may communicate with a periapical inflammatory lesion microscopically diagnosed as an abscess, granuloma or cyst.
- (6) Only 2 of the 15 biopsied periapical lesions had a radiolucency smaller than 5 mm. in diameter. They were both over 3 mm. in diameter.

INFORMED CONSENT

"The informed consent of all human subjects who participated in the experimental investigation reported in this manuscript was obtained after the nature of the procedures and possible discomforts and risks had been fully explained."

"The opinions expressed herein are those of the authors and are not to be construed as those of the Department of the Army or the Department of Defense."

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Table 1. Microscopic examination of 15 sinus tracts without the periapical lesion

	Sinus tract lining	Epithelium	Granulomatous inflammatory tissue	Total
	Number of specimens	5	10	15
※	1		1	 -1
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Table 2. Microscopic examination of 15 sinus tracts and the periapical lesions from which they originated

Patient	Tooth number	Position of tract	Size of radiolucency	Microscopic periapical diagnosis	Epithelial lining in sinus tract
1	5	facial surface (F)	12 x 15 mm.	cyst	present (+)
2	18	F	4 x 5 mm.	granuloma	absent (-)
3	14	palatal surface (P)	3 x 4 mm.	granuloma	+
4	4	F	6 x 7 mm.	granuloma	-
5	18	F	6 x 7 mm.	abscess	-
6	19	F	5 x 5 mm.	abscess	+
7	30	F	5 x 7 mm.	abscess	-
8	14	F	5 x 8 mm.	cyst	-
9	3	F	3 x 4 mm.	abscess	+
10	30	F	5 x 5 mm.	abscess	-
11	5	F	7 x 8 mm.	abscess	-
12	30	F	5 x 6 mm.	abscess	-
13	7	F	5 x 5 mm.	granuloma	•
14	3	F	5 x 7 mm.	cyst	+
15	19	F	7 x 10 mm.	cyst	-

FIGURE LEGEND

Fig 1. Cross-sectional views of epithelium lined sinus tract at different levels.

Level A: Section through sinus stoma.

Level B: Section at epithelial rete ridge-connective tissue interface.

Level C: Section near periapical level.

- Fig 2. Transverse section through sinus tract at level of surface mucosa showing complete development by epithelium.

 (Hemotoxylin and eosin stain. Magnification X40)
- Fig 3. Transverse section at lower level of rete ridges. Black arrow points to sinus tract epithelium. Outline arrow points to rete ridge epithelium. (Hemotoxylin and eosin stain.

 Magnification X40)
- Fig 4. Transverse section near periapex showing sinus tract epithelium surrounded by connective tissue infiltrated with inflammatory cells. (Hemotoxylin and eosin stain. Magnification X40)
- Fig 5. A sinus tract with an epithelial lining of the surface orifice.
- Fig 6. A sinus tract with an epithelial lining extending to the periapical area.
- Fig 7. A sinus tract with an epithelial lining that merges with the lining of a periapical cyst.
- Fig 8. A sinus tract with epithelium at the surface orifice and additional epithelium proliferating into the tract from a periapical cyst.



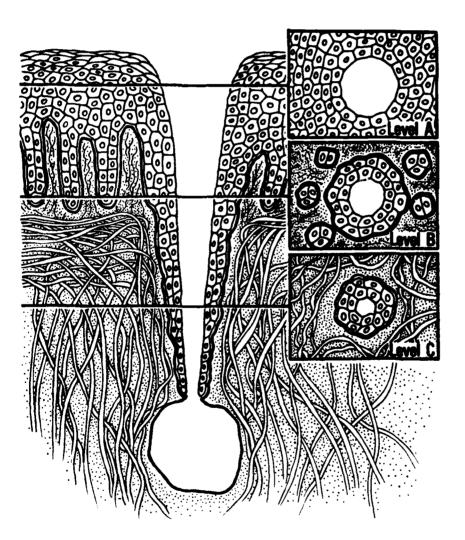


FIGURE 2

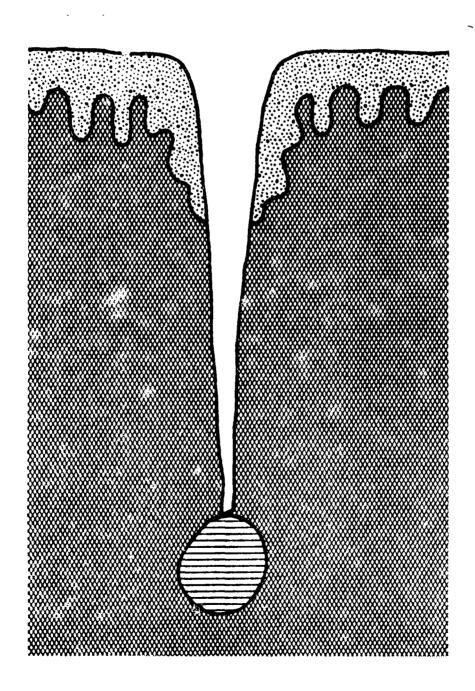


FIGURE 3

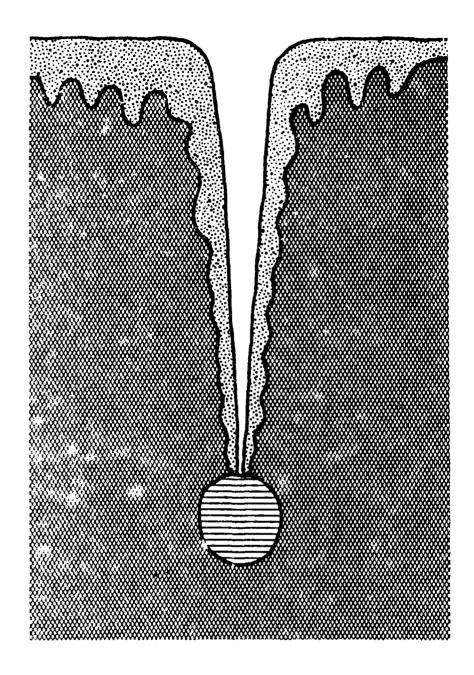


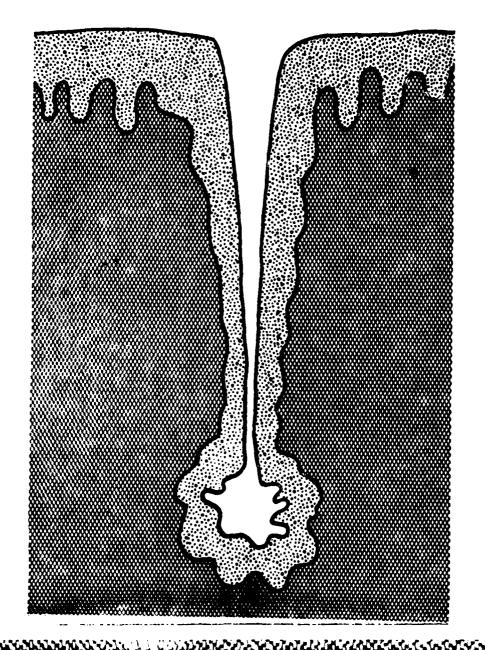


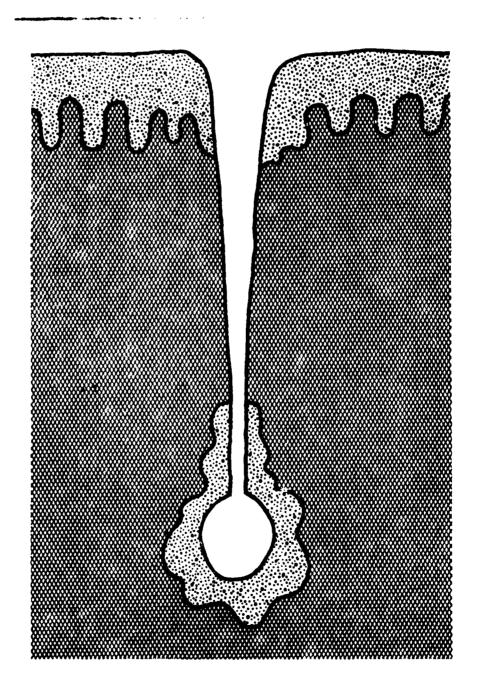












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